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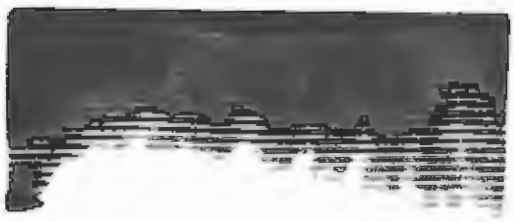
TRANSILLUMINATED BIOMICROSCOPY AND INFRARED PHOTOGRAPHY OF IN-VIVO MEIBOMIAN GLAND MORPHOLOGY



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Introduction:
Chronic lid margin disease is often referred to as anterior lid margin disease (seborrheic and staphylococcal blepharitis) and posterior lid margin disease (meibomian gland dysfunction). Meibomian gland dysfunction (MGD) has been used to describe the spectrum of clinically observed meibomian gland abnormalities previously described as meibomitis. However, the lack of a consistent glandular inflammatory response makes meibomian gland dysfunction the more appropriate term.[20]

MGD is a common and perplexing condition with an obscure etiology. It is believed to be a congestion of the meibomian glands with a chronic stagnation of meibum (meibomian gland secretion). The condition appears to have a combined staphylococcal, immunologic, and mechanical etiology.[4] The spectrum of MGD can range from minimal clinical significance to sight-threatening ocular rosacea. In addition, MGD has been suggested as an important factor in the pathogenesis of several ocular conditions, including chronic blepharitis, hordeola, chalazia, and even contact lens intolerance.[10,22] To better understand the mechanisms and clinical manifestations of MGD, it is first necessary to review the anatomy and physiology of the various sebaceous and sweat glands on the external or skin side of the upper and lower lids.

GLANDS OF MOLL

The glands of Moll, located at the root of the eyelashes, are considered sweat glands that have become arrested in their development. The glands were first described by Jacob Anton Moll in 1857. The body of the gland forms an oval or spherical ball, and the superficial portion forms the duct that opens onto the lid margin in the spaces between two lashes.[3] The ducts may also empty into the Zeis glands or into the follicles of the cilia close to the surface. The glands are more numerous in the lower lid, although there is not one to every lash.

GLANDS OF ZEIS

The glands of Zeis are modified sebaceous glands which are attached directly to the follicles of the eyelashes. There are usually two glands per cilium. The gland sebum passes out between the lash and its epithelial covering. The purpose of this oily secretion, as elsewhere in the body, is to prevent the hair (lash) from becoming dry and brittle.[3,23]

THE MEIBOMIAN GLANDS

The tarsal glands were first adequately described by Meibomius in 1666.[3] They are enormously developed sebaceous glands that belong to a category of holocrine glands whose secretions are composed of entire cells that are released upon maturation.[10] The meibomian glands are arranged vertically parallel with each other, numbering approximately 25 in the upper lid and 20 in the lower lid.[23] The glands of the upper lid are larger than those in the lower lid, owing to the larger size of the upper tarsal plate.[3]

Each gland consists of a central canal, into the sides of which open many acini, rounded lateral

ducts, numbering 30-40 in the larger glands. The glands are closed at the inner end, and at their outer end lies an excretory duct. Each excretory duct is lined by four layers of squamous epithelial cells situated on a basement membrane. This epithelial layer increases to six cells as the duct approaches the lid margin. The keratinization of these squamous ductile cells is responsible for much of the obstruction of the glands in MGD. The duct mouth opens to form small orifices at the margin of the lid on the inner side of its posterior border. The glands are surrounded by lymph spaces and are supplied by nerves and blood vessels which traverse the tarsus to reach them.[3]

The meibomian glands are sebaceous in nature. Their secretion, called meibum, originates in the most remote part of the gland within the acini where the cell's cytoplasm accumulates minute droplets of sebaceous material, resulting in the characteristic foamy appearance on gland expression. As the cell reaches full maturity the nucleus shrinks and the membrane wall ruptures, releasing all of its contents.[10] The sebaceous secretion is expelled into the excretory duct, where it mixes with the desquamated epithelial cells from the duct lining.[5] The viscous meibum collected within the excretory duct is then delivered to the tear film through the orifices at the lid margin. Owing to the location of the glands, between the tarsal plates in each lid, the simple act of blinking is sufficient for the expression of the meibomian secretion into the tear film. Andrews[1] has observed that the spreading of the thick meibum on the aqueous surface of the tears may be accomplished by the presence of a surfactant or solvent component in either the meibum or the tear film.

The major functions of meibum in the tear film are

- To provide lubrication between the lid margin and the cornea
- To ensure an airtight closure of the lids
- To prevent the tears from macerating the skin
- To supply a limiting hydrophobic oily film for the tear film to retard evaporation

LACRIMAL GLANDS

Opening into the conjunctival side of the lids are the main lacrimal gland and accessory glands of Wolfring (two to four glands in the upper lid and one to two in the lower lid) and Krause (42 in the upper lid and six to eight in the lower lid). Collectively, these exocrine glands form the intermediate or middle aqueous layer of the precorneal tear film. The mucin-secreting goblet cells are diffusely distributed throughout the conjunctiva, including the crypts of Henle. These cells are composed of high molecular weight glycoprotiens and other protein components.[22]

BIOCHEMISTRY OF MEIBUM

Throughout the years a number of researchers have performed analytical testing of human meibomian secretions. Early investigations by Pez (1897) and by Linton, Curnow, and Riley (1961) were largely qualitative, with only rough indications of the relative magnitudes of lipid components.[21] In the 1960's, further biochemical analyses by thin-layer and gas-liquid chromatography revealed a more complete picture of the makeup of human meibum.[8,18] Today, meibum is generally considered to be a combination of wax esters, cholesteryl esters, triglycerides, fatty acids, hydrocarbons, fatty alcohols, cholesterol, and phospholipids.[22] There appear to be

great variations in the relative makeup of individual meibum components. For this reason, Tiffany[21] concluded that no "typical" composition for human meibomain oil exists and that "mean" values are meaningless. Therefore, the synthesis mixing of an artificial tear based on the "average" meibum composition would not be desirable.[21]

MEIBOMIAN GLAND DYSFUNCTION

As previously discussed, it is generally accepted that chronic lid margin disease can be divided into anterior lid margin diseases (seborrhea, staphylococcal) and posterior lid margin disease (meibomian gland dysfunction). Meibomian gland dysfunction can be simply defined as physical obstruction of the glands by plugging of the excretory ducts. The blockage causes stagnation of the gland secretions, initiating a wide variety of clinical complications.

The mechanism for this blockage appears to be located at the level of the epithelium lining the excretory duct. The excretory duct of the meibomian glands is lined by the same type of epithelium as the surface of the skin. Therefore, excretory duct obstruction could occur under circumstances of increased epithelial turnover in which cells are detached in the form of small scales, similar to dandruff, or abnormal keratization or hyperkeratinization of the duct epithelium (including sloughing of keratin and narrowing of the duct), or both.

Increased Epithelial Turnover

Certain dermatologic conditions, such as seborrheic dermatitis and acne rosacea, are characterized by increased epidermal turnover. Large amounts of cells are produced that detach from the epidermal surface in the form of small scales. The mechanism is similar to that of dandruff production.[10] Therefore, it is reasonable to assume that duct obstruction is more likely to occur under circumstances of increased epithelial turnover, owing to accumulation of desquamated epithelial cells.

Keratinization of the Meibomian Gland Duct

Recent studies have indicated that MGD may result from hyperkeratinization of the duct epithelium.[20] This keratinization does not end at the orifice but extends more posteriorly throughout the gland duct.[7] In a histopathologic study of seven patients with MGD, Gutgesell et al found signs of obstruction and dilation of the meibomian gland ducts, along with acinar enlargement and hyperkeratinization of the duct epithelium.

These changes are not always accompanied by pouting of the orifices or inflammatory signs along the lid margin, and the condition is often overlooked. Under these circumstances, however, expression of the gland often releases an inspissated material. This, combined with the patient symptoms, are critical in making the diagnosis of MGD. Observations concerning MGD and keratinization of the meibomian gland may be the counterpart to the well-recognized relationship between skin disease and sebaceous gland dysfunction. Knutson[9] has shown that keratinization of

the sebaceous gland duct leading into the pilosebaceous canal is the critical step in the development of clinical acne vulgaris.

Physical obstruction of the meibomian gland ducts (either partial or total) results in two complications, stagnation and possible infection.[10]

Gland Stagnation

Studies on meibomain gland composition have shown that the meibomian lipids in patients with MGD do not differ significantly from those of normal subjects.[19] It appears that gland health and structure are unaffected by the meibum makeup in MGD. Gutgesell et al[4] concluded that stagnation of meibum causes an increase in pressure within the gland and that this factor may be responsible for the wide variety of histologic changes seen in MGD. Therefore, the health of the meibomian glands may be best determined by evaluating glandular morphology rather than meibum composition.

TRANSILLUMINATED BIOMICROSCOPY

In vivo gland structure is best viewed through a modified slit-lamp technique, using a standard transillumination light source. The patient's lower lid is gently everted over the transillumination light probe, which allows direct visualization of the meibomian glands through the palpebral conjunctiva.[17,20] The gland morphology can be photographically documented with high-speed infrared film. This technique appears to be less subjective and provides a greater degree of glandular detail than standard slit-lamp examination.

Materials and Methods:
Transilluminated meibomian gland slit-lamp infrared photography was performed on 62 subjects ranging from 21to 45 years of age, and ranging from zero meibomian gland dysfunction symptoms to severe symptoms. All subjects that participated in this study were students at Pacific University College of Optometry. Approximately half of the subjects were current contact lens wearers.

A Nikon MF-12 35mm camera was attached to a Mentor slit-lamp. From previous studies of meibomian gland photography[20], high-speed infrared Kodak film with a setting of f/32 at 1/60 sec. was shown to provide optimal meibomian gland appearance. With the Nikon camera used in this study, there was no setting to control the f-stop. Exposure time was set at 400 ASA and the camera was set to B&W (black and white). Loading and unloading of the film was performed in total darkness due to the qualities of infrared film. Infrared film was used due to it's ability to pick up differences in heat gradients.

The Alcon transilluminator was used with a fiber optic probe to evert the lower lid and illuminate the structures of the meibomian glands. The sole source of light came from the probe, therefore no camera flash or room illumination was needed. Once the probe was in place, the everted lower lid was focused with the biomicroscope. After which, the biomicroscope was turned down to emit no light. Photographs were taken with refocusing after every two shots, with a total of four

shots per eye (see figure 1 and 2).

Results:
The methods used produced crisp clear photographs of the meibomian gland structures. Photographs show that different changes can occur within the meibomian glands that are not noticeable with standard biomicroscopy techniques. This alone shows a need for this procedure.

Classification of Meibomian Gland Dysfunction

Clinical classification of MGD is difficult because of the diversity of morphologic and subjective features of the condition. In addition, there is frequent association of MGD with other underlying dermatologic conditions (e.g., acne rosacea, staphylococcal blepharitis). These must be considered when patients are evaluated for MGD.

The following classification system of Caroline and Kame[2] is based on objective changes in the meibomian glands as viewed through the conjunctiva of the everted lower lid and the orifices of the glands located at the lower lid margin. The tear meniscus is viewed at the junction between the lower lid and the globe, and the bulbar and tarsal conjunctiva are examined throughout by standard biomicroscopy techniques.

Grade 0 MGD, Normal

Signs

Meibomian Glands. Glands are uniform with a "piano key" appearance. No engorgement or stagnation of gland structure is present. Glands may not be visible through the lower lid conjunctiva.
Meibomian Gland Orifices. Orifices appear as tiny holes or only potential openings, or may not be seen at all. No pouting or plugging is present; clear meibum can be seen on gentle expression.
Tear Film. No foaming is observed, and the tear meniscus is normal.
Conjunctiva. No congestion or injection are observed.

Symptoms

No symptoms are usually present.

Grade 1 MGD, Minimal

Signs

Meibomian Glands. Some glands appear normal; others appear engorged, widened with early stagnation of meibum. More prominent changes occur near the orifices.
Meibomian Gland Orifices. The orifices are more obvious, with some pouting and early plugging. There may be an abundance of clear oily excretion on gentle expression.

Tear Film. The tear film is clear but may have some foaming in the central areas of the meniscus.
Conjunctiva. Trace congestion and injection are present. There may be some mild papillary hypertrophy with mild rose bengal staining.

Symptoms

The patient may be asymptomatic or may experience a nonspecific foreign-body sensation (greater in the morning). There may be transient symptoms of a burning sensation and dryness.

Grade 2 MGD, Mild

Signs

Meibomian Glands. Many glands are dilated and engorged, with obvious stagnation of meibum. There is increased irregularity of gland shape, especially near the lid margin. Subconjunctivally, there are discrete collections of lipid (not solid and not microchalazia). The patient may have one to three "true" microchalazia per lid, which appear as dark round spots on the photographs.
Meibomian Gland Orifices. Most orifices are obvious and slightly dilated with pouting and plugs. There may be mild inflammation around the orifices. The glands contain an abundance of clear oily material, which may appear whitish-yellow on expression.
Tear Film. The tear film may be clear with some foam present at the canthus.
Conjunctiva. Mild congestion and injection with increased papillary changes and rose bengal staining are observed.

Symptoms

Symptoms may be minimal or absent. The patient may experience slight tearing, foreign-body sensation, irritation, burning, or itching, and may report red eyes with mattering or crusting along the lid margins. Symptoms of transient ocular dryness are common.

Grade 3 MGD, Moderate

Signs

Meibomian Glands. There may be gross enlargement of many glands secondary to meibum stagnation. Multiple collections of lipid may be present subconjunctivally (not microchalazia), with three or more microchalazia in the lower lid.
Meibomian Gland Orifices. These may exhibit increased orifice pouting, and plugging, with inspissation in most. Gland expression is difficult and is often preceded by the extrution of

an inspissated plug, followed by whitish-yellow or abundant clear oily material. Lid margins may be irregular, edematous, and inflamed.
Tear Film. Tear film debris may be present along the lower lid margin. Foam may be present centrally and at the inner or outer canthus. There is a rapid tear break-up time.
Conjunctiva. Moderate congestion and injection overlie the affected glands. There are moderate papillary hypertrophic changes, with increased rose bengal staining.

Symptoms

Moderate irritation, foreign-body sensation, tearing, mattering, redness, burning, and itching may be present, with increasing symptoms of ocular dryness. The patient may have cosmetic concerns because of the red rim around the eyes, and may have a history of chalazia.

Grade 4 MGD, Severe

Signs

Meibomian Glands. Numerous collections of lipid with increased numbers of microchalazia are present, and the patient may have frank chalazion.
Meibomian Gland Orifices. There may be increased pouting, plugging, and inspissation. Gland expression is difficult due to near complete blockage. Meibum may be an abundant clear oil or whitish-yellow material. The lid margins are irregular, edematous, and inflamed.
Tear Film. There is obvious foamy discharge in the tear film, with excessive debris and rapid tear break-up time.
Conjunctiva. There is moderate to severe congestion and injection, with increased papillary hypertrophy.

Symptoms

The patient has chronic symptoms of irritation, redness, discharge, foreign-body sensation, tearing, burning, and itching. There are chronic symptoms of ocular dryness, and often a history of recurrent chalazia.

SIGNS AND SYMPTOMS OF MGD

The possibility of MGD is usually not investigated unless the patient presents with significant symptoms or gross signs.[5] The signs and symptoms of MGD are usually bilateral and demonstrate little asymmetry.[15] The lid margins may be normal in the early stages of MGD. In the later stages they become thickened and rounded, with telangiectatic blood vessels crossing the lid margins.

Patient symptoms may not be present unless the integrity of the tear film is stressed by changes in humidity and temperature or by the presence of a contact lens. The stagnation of the meibomian glands may account for the tear film instability. This is suggested by the fact that stabilization of the tear film occurs when fresh secretions from deep within the glands are added by expressing them directly into the tear film.[15] The observed instability cannot be ascribed to a quantitative

decrease in the tear lipid layer, since these patients have normal or increased interference patterns in the tear film, indicating that no shortage of lipid exists.[16]

MGD may or may not present with ocular surface involvement in the form of SPK, which is greatest in the intrapalpebral space. The SPK has the appearance of that seen in other conditions secondary to an unstable tear film. It is *not* similar to the keratitis that has been described secondary to *Staphylococcus* toxin, which is more typically seen in association with anterior bacterial blepharitis. The finding of an unstable tear film in patients with posterior blepharitis most likely accounts for this keratopathy without having to postulate the presence of a bacterial exotoxin.[15] Studies by Korb et al[10] found an incidence of corneal staining in 41 (57.7%) of symptomatic eyes (possible MGD) and in only eight (10 percent) of control (normal) eyes. The corneal and conjunctival superficial punctate staining may not be seen with the initial instillation of a single drop of 2 percent liquid fluorescein. However, a sequential drop of fluorescein instilled at 5-minute intervals for 20 minutes may reveal an underlying epitheliopathy.[11]

McCulley and Sciallis[15] examined the TBUT in patients with MGD and noted a breakup time of 9 seconds or less (mean 7.18 ± 0.42 seconds). This compares to a mean TBUT of 28.22 ± 0.43 seconds in normal control patients. It was observed that when the TBUT was repeated immediately after expression of the meibomian glands, it returned to normal or supernormal levels in all MGD patients (mean 29.35 ± 2.85 seconds).[15]

Another common tear film finding in MGD is the presence of foamy tears. Andrews[1] has suggested that the frothy secretion may be related to an overproduction of irritative fatty acids secondary to the lipid infection and/or inflammation. Korb and Henriquez[10] reported the finding of foamy tear film at the lower lid margin or the lateral canthus in 47 (66.2 percent) eyes with MGD and in only three (3.7 percent) control eyes

In the more advanced stages of MGD, small (2-3mm) microchalazia can be noted within the meibomian glands. The exact histopathologic composition of these spots in unknown, but the presence of a microchalazion may be the earliest change leading to a frank chalazion. They often present 1 to 2 mm posterior to the lid margin on the conjunctival side. These must be distinguished from concretions, which are inclusions within cystic structures in the conjunctiva, whereas microchalazia are deeper and have a mounded configuration.

GLAND EXPRESSION

Under normal circumstances the meibomian glands secrete a clear viscous oil with gentle digital pressure on the lower lid. However, in MGD the sebaceous material and cell debris from within the meibomian gland and the duct epithelium become cloudy or absent on expression. Inspissated secretions may appear as

- Very fine, translucent filamentary secretion
- Secretion in column form (toothpaste)
- Creamy secretion (pus-like)

Inspissated secretions can be revealed only by more aggressive pressure directed on the lower lid.

Aggressive gland expression can be performed by pressing a rounded glass or plastic rod upward against the lid towards the globe.[12] In addition, the glands can be expressed by squeezing (pinching) the lower lid between the thumb and forefinger.

Korb and Henriquez[10] described an additional technique for forceful gland expression, which begins with the instillation of 2 percent proparacaine into the inferior cul-de-sac. Expression is performed by compressing the lower lid with moderate force between a sterile cotton swab on the palpebral conjunctival surface, and the thumb on the side of the skin. A similar technique of gland expression is performed by massaging the lid between two sterile swabs.[15]

Studies by Norn found that in apparently normal subjects only 44 percent of the meibomian glands were discharging secretions into the tear film at a particular time. On the basis of this data, it appears that very little gland secretion is necessary to allow normal lipid tear film synergy.[6]

Studies by Korb and Henriquez found a high incidence of absent secretion on gentle expression in 26 (36.6 percent) of symptomatic, possibly MGD eyes. Secretion was absent in only two (2.5 percent) of control "normal" eyes.[10]

BACTERIOLOGY

The dead and desquamated epithelial cells within the stagnated secretions of the meibomian glands may provide an excellent culture medium for bacteria and represent a continuous source of irritation to the ocular surface.[10]

Bacteriological studies by Korb and Henriquez[10] indicate that the most frequent organism identified in MGD are *Staphylococcus epidermidis* and *S. aureus.* Studies by McCulley and Sciallis[16] on 43 patients with MGD found 42 percent positive for *S. epidermidis.* Thirty-seven percent grew *S. aureus*, 10 percent diptheriods, and 22 percent grew *Proprionibacterium acnes.*[16]

McCulley and Dougherty[13] also reported that expression of the meibomian glands in MGD patients always reflected the bacteria found along the anterior lid margin. However, the recovery rate of the glandular (meibum) bacteria was considerably less than the recovery rate from the anterior lid margin. Therefore, MGD may not represent a primary infectious disease but instead may reflect the contamination of the meibomian secretion from the lid margins at the time of obtaining the cultures.[13]

McCulley et al[14] reported that meibum analysis has failed to reveal a qualitative abnormality in patients with MGD. However, preliminary gas-liquid chromatography data on the secretions suggests an increase in free fatty acids and a shift towards lipids with higher melting points. The free fatty acid not only could destabilize the tear film but could cause an epithelial abnormality as a result of direct toxicity to the cells. Lipids with higher melting points would tend to stagnate the secretory flow, allowing greater access by bacterial lipolytic exoenzymes to the stagnant lipid pool that subsequently forms the lipid layer of the tear film. This could account for the fact that no single pathogen has been found in these patients, and provides at least an impossible mechanism by which bacteria contribute to the inflammatory disease process.[14]

MGD TREATMENT

Practitioners must explain to patients that chronic MGD is a condition that has no cure. It should be stressed that initial (and relatively vigorous) therapy is prescribed to bring the condition under control. After this acute phase of therapy, a minimal (variable) amount of chronic treatment will be required to maintain control.[14]

Treatment of MGD should be directed towards relieving the obstruction of the ducts and orifices, to allow normal flow of the meibomian gland secretions onto the precorneal tear film.[5] This is best accomplished by

1. Treatment of anterior seborrheic staphylococcal blepharitis (if present)
2. Hot compresses bid to qid
3. Lid scrubs bid to qid
4. Forceful expression of the meibomian glands
5. Topical antibiotics (if necessary)
6. Tetracycline 250 mg PO qid (on an empty stomach) for 10 to 30 days

The following approach to instructing patients on home hygiene and therapy for MGD is recommended. Patients should be instructed to use a warm compress by placing a facecloth under hot running water. The cloth should be as warm as can be tolerated (not scalding) and applied to the closed lids for 5 to 10 minutes. The facecloth should be repeatedly rewetted to maintain it at a suitable warm temperature. The patient is then instructed to place baby shampoo (undiluted or 50 percent diluted with water) on a moist face cloth and to scrub the lids and lashes with horizontal strokes. The patient should be told that the major goal of this maneuver is to remove debris from the base of the lashes. This is facilitated by the prior application of warm compresses that loosen and partially melt the debris along the lid margins. After the warm compresses and lid scrubs, the patient is instructed to wash away the excess shampoo. Patients are instructed to perform this procedure two to four times a day, depending on the amount of debris present and the degree of inflammation. It should be stressed that the most important time to apply the warm compresses and lid scrubs is in the morning, after the debris has collected and stagnated overnight.

Expression of the meibomian glands should be performed professionally in the office at appropriate intervals. In addition, patients should be carefully instructed on home therapy, consisting of lid massage after warm compresses and before the lid scrubs. This maneuver is best accomplished by taking the tip of the finger and placing against the globe using small, circular motions. This should be performed along the entire course of the meibomian glands, massaging both the upper and lower lids.

On initial examination of patients with MGD it is often difficult to determine if antibiotic therapy is necessary. If there is evidence of staphylococcal blepharitis, a topical antibiotic ointment should be prescribed. The ointment should be applied to the lids after local hygienic maneuvers. Patients are instructed to place a small amount of ointment on a clean fingertip and rub the antibiotic into the lids and lashes. This is done two to four times a day, depending on the severity of the inflammatory process. If there is significant associated keratoconjunctivitis, an antibiotic drop can

be prescribed four times a day.

McCulley and Sciallis[15] and Gutgesell et al[4] have found only a minimal and inconsistent increase in the inflammatory cells in the glands of patients with MGD. Therefore, corticosteroids and other inflammatory agents are unlikely to modify the course of the condition and should be discouraged.

Studies have indicated that many patients with MGD respond well to oral tetracycline antibiotic therapy. The exact mechanism of this drug action is not known. Gutgesell et al[4] suggest that the drug reduces the bacterial population and thus the quantity of lipolytic enzymes. This action may decrease the amount of free fatty acid in the meibomian glands.

Most patients with grade 3 or greater MGD should be started on oral tetracycline along with the local lid hygiene maneuvers. The initial dose is oral tetracycline 250 mg qid to be taken on an empty stomach and continued until the MGD is under control, after which time it can be slowly tapered. The majority of patients can be tapered from the tetracycline within 4 to 12 weeks; however, an occasional patient will require low-dose oral tetracycline indefinitely to maintain control.

Intensive, acute therapy should be maintained until the blepharitis is under control. This typically requires 2 to 6 weeks of vigorous therapy, after which the intensity of the therapy can be decreased. The goal should then be to determine the minimal amount of maintenance therapy required to maintain control. This frequently entails warm compresses and lid scrubs once or twice a day. An attempt should be made to avoid the chronic use of antimicrobial agents, either topically or systemically.

The use of preservative-free artificial tears for patients with concurrent keratoconjunctivitis sicca is also recommended.

Further testing will be needed to quantify the number of patients that fall into the different categories and to find a photographic match for these categories.

Summary:

The use of transilluminated meibomian gland photography has proved to be a relatively simple in-office technique that allows accurate and detailed information about the meibomian gland structures. There are many problems that arise due to meibomian gland congestion, but quantifying the level of dysfunction has always varied from practioner to practioner. The grading scale along with the outline for photography of the glands allows for a more repeatable grading system. The ease and benefit of the procedure should make this technique as second nature as posterior segment photography.

The new technique for photography of in-vivo meibomian gland morphology is not going to be the only test used to diagnosis MGD, but it would add another dimension to the grading and documentation. The photograph's along with slit-lamp signs, case history, and tear break-up time would give the practioner a thorough exam to accurately diagnosis and grade meibomian gland dysfunction.

There needs to be further research done to quantify the number of people in each of the categories and to find photographic matches to be included with the five written grades. That would allow the practioner to have photographic matches to an already detailed grading scale.

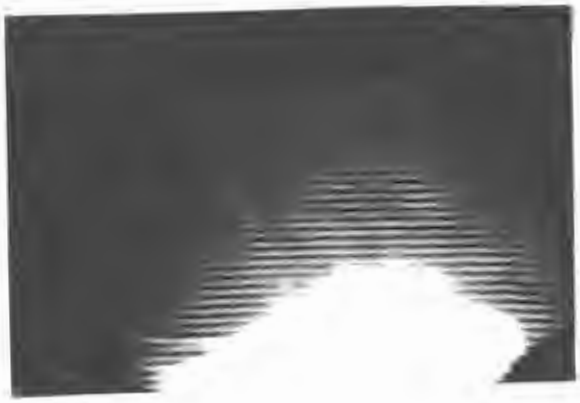

Figure 1

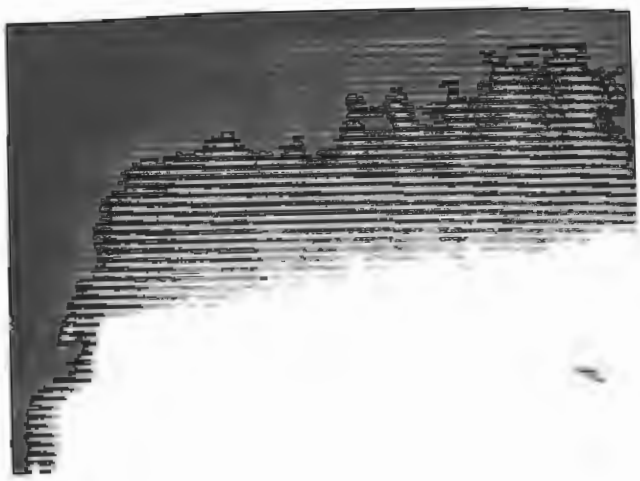

Figure 2

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